OVERALL SUMMARY FOR FLUOROBENZENE

Summary

Fluorobenzene is a liquid with a water solubility of approximately 1540 mg/L. Fluorobenzene has a freezing point of -40°C, boiling point of 84.73°C at 760 mm Hg, density of 1.024 g/mL, and a vapor pressure of 100 mm Hg at 30.4°C.

A review of estimated physical-chemical properties and environmental-fate characteristics indicates that fluorobenzene may be persistent in air with an estimated half-life due to hydroxyl radical oxidation of 23.3 days. Based on the BIOWIN ultimate survey model estimate of weeks-to-months, fluorobenzene may be moderately persistent in terrestrial compartments, and is not expected to readily biodegrade (Table 1). Fluorobenzene is not highly bioaccumulative with an estimated BCF of 11.17 (Table 1). When modeled using a Level III fugacity model under a standard scenario of equal emissions to air, water, and soil, fluorobenzene is expected to partition primarily into air and water compartments (Table 1). The Hydrowin model (v. 1.67, Syracuse Research Corporation) could not estimate a hydrolysis rate for fluorobenzene in regard to stability in water. However, halogenated aromatics/PCBs are generally resistant to hydrolysis (Harris, 1990), and thus fluorobenzene would be likely to be stable to hydrolysis in water. A hydrolysis test using OECD Guideline 111 is recommended to confirm this prediction.

Table 1 : Environmental Fate

Bioconcentration*	BCF = 11.17
Biodegradation*	Does not readily biodegrade
Fugacity*	Level III Partition Estimate Air 40.9 % Water 44 % Soil 14.8 % Sediment 0.245 %
* Modeled data	

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In aquatic organisms, fluorobenzene has low toxicity to fish with a 96-hour LC_{50} in fathead minnows of 210 mg/L. Fluorobenzene was moderately toxic to *Daphnia* in a 24-hour study which produced an EC_{50} of 7.37 mg/L. Modeling of physical-chemical parameters (i.e., log Kow) and aquatic toxicity was conducted to help provide insight into the behavior in the environment and the aquatic toxicity of fluorobenzene (See Table 2). Syracuse Research Corporation models for estimating physical-chemical properties were used to estimate $\log_{10}$ Kow (Meylan and Howard, 1995) for subsequent use in the ECOSAR program. ECOSAR (Meylan and Howard, 1999) was used to estimate aquatic toxicity data for green algae, daphnids (planktonic freshwater crustaceans), and fish. ECOSAR predictions are based on actual toxicity

test data for classes of compounds with similar modes of action. The predicted $\log_{10}$ Kow value was used as input for the ECOSAR model (see Table 2 for values). The ECOSAR predictions indicate that fluorobenzene is of low to medium concern relative to acute toxicity to algae, invertebrates, and fish.

Additional aquatic toxicity data are presented for another mono-substituted halobenzene (chlorobenzene) as well as several di-substituted halobenzenes (chloro-fluorobenzenes). The acute fish, daphnid, and algae data for chlorobenzene support the data presented for fluorobenzene. Although the fluorobenzene and chlorobenzene acute test data for daphnids are from 24-hour tests, the 48-hour chloro-fluorobenzene test data (based on measured test concentrations) support the data for the monosubstituted halobenzenes. The dichlorobenzenes have been reported to be more toxic than monochlorobenzene (Galassi and Vigli, 1981) although position of the chloro substituted groups on the benzene ring appears to have no affect on aquatic toxicity (US EPA, 1980). This pattern also appears to hold true for the chloro-fluorobenzenes since they appear to be more toxic than either chlorobenzene or fluorobenzene and the position of the fluoro groups on the benzene ring has no effect on aquatic toxicity. ECOSAR appears to underestimate the toxicity of these compounds to daphnids, but the experimental data are adequate for acute hazard assessment. The existing data (experimental and estimated) are also adequate for assessing acute hazard to fish and algae, therefore no additional testing is necessary.

Table 2: Aquatic Toxicity Values

	Fluorobenzene	Chlorobenzene	1-Chloro-2-fluorobenzene	1-Chloro-3-fluorobenzene	1-Chloro-4-fluorobenzene
Log Kow	2.19	2.64	2.84	2.84	2.84
Toxicity to Fish (LC_{50} value)	96-hour: 210 mg/L (N) 48-hour: 430.5 mg/L (N) 96-hour: 47.2 mg/L (E)	96-hour: 10.4 mg/L (N) 96-hour: 20.9 mg/L (E)	No test data. 96-hour: 15.7 mg/L (E)	No test data. 96-hour: 15.7 mg/L (E)	No test data. 96-hour: 15.7 mg/L (E)
Toxicity to Invertebrates (EC_{50} value)	24-hour: 7.37 mg/L (M) 48-hour: 51.3 mg/L (E)	24-hour: 4.3 mg/L (N) 48-hour: 23.4 mg/L (E)	48-hour: 2.28 mg/L (M) 48-hour: 17.8 mg/L (E)	48-hour: 3.64 mg/L (M) 48-hour: 17.8 mg/L (E)	48-hour: 1.70 mg/L (M) 48-hour: 17.8 mg/L (E)
Toxicity to Algae (EC_{50} value)	No test data. 96-hour: 32.4 mg/L (E)	96-hour: 12.5 mg/L (N) 96-hour: 15.2 mg/L (E)	No test data. 96-hour: 11.7 mg/L (E)	No test data. 96-hour: 11.7 mg/L (E)	No test data. 96-hour: 11.7 mg/L (E)
E = estimated value, N = value based on nominal test concentrations. M = value based on measured test concentrations.					

Fluorobenzene has very low acute oral toxicity with an acute lethal dose (ALD) > 11,000 mg/kg in rats. No clinical signs of toxicity were observed in the non-lethal doses; however, slight to severe weight loss was noted. Fluorobenzene also had very low acute inhalation toxicity with a 4-hour acute lethal concentration (ALC) of 6200 ppm in rats. When applied to the skin of rabbits, fluorobenzene produced no to mild erythema and no to severe edema and was considered a mild skin irritant. Fluorobenzene did not induce dermal sensitization in guinea pigs. Fluorobenzene was moderately irritating to the rabbit eye in one study, and severely irritating in two other studies containing limited information. More severe effects were observed in eyes that were washed than those that remained unwashed.

In the following sections, subchronic studies on fluorobenzene and chlorobenzene are presented to support the fluorobenzene toxicity database. Developmental, reproductive, and chronic studies are available only for chlorobenzene. Chlorobenzene should be an acceptable structurally similar analog to support the HPV database for fluorobenzene for the following reasons. The metabolism of monohalobenzenes in mammals has been well studied. Qualitatively, monohalobenzenes, including chloro-, bromo-, iodo-, and fluorobenzene are all metabolized by common metabolic pathways in mammals. All halobenzenes are initially oxidized to the corresponding halophenol, either directly or via an intermediate epoxide. The epoxide intermediates may be hydrolyzed to dihydrodiols which are in turn oxidized to catechols, or may undergo conjugation with glutathione, resulting in the excretion of the corresponding mercapturic acid in the urine. A more detailed discussion of the metabolism of halobenzenes is presented at the end of the toxicity section. Overall, the pathways for metabolism of halobenzenes, including chlorobenzene and fluorobenzene are essentially identical. Thus chlorobenzene toxicity studies should be acceptable to support the fluorobenzene HPV database.

In a 28-day repeated dose study, groups of rats were exposed nose-only, 6 hours a day to 0.4 mg/L (94 ppm), 1.5 mg/L (381 ppm), or 6.0 mg/L (1585 ppm) of fluorobenzene. Slight changes in physical condition were seen for rats exposed to 1.50 or 6.24 mg/L. Other effects of treatment were confined to adaptive liver changes and unique male rat hydrocarbon nephropathy. Although the adaptive liver changes extended into the low dose group (0.37 mg/L), neither of these conditions were considered to be indicative of toxicologically important adverse effects of treatment and, consequently, the NOAEL was considered to be 0.37 mg/L (94 ppm). Furthermore, the slight changes observed in physical condition were not indicative of serious damage to the health of the animals. There was, however, evidence of a treatment-related increase in fluoride concentration in bones and teeth of animals from all exposure groups.

In a 13-week subchronic study, groups of ten male and ten female Fisher 344 rats or B6C3F1 mice were dosed with 0, 60, 125, 250, 500 or 750 mg/kg/day chlorobenzene. The NOEL was 125 mg/kg/day. Effects observed at higher doses in rats included reduced body weight gain, reduced survival, clinical chemistry changes, organ weight changes, and pathological changes in liver, kidney, thymus, bone marrow, and spleen. In a 13-week inhalation study, 15 Charles River rats/gender/group and 4 beagle dogs/gender/group were exposed to 0, 0.75, 1.5, or 2.0 mg/L chlorobenzene 6 hours/day, 5 days/week. Effects observed in rats at 0.75 mg/L or higher included hypoactivity, reduced body weight, and organ weight changes. There were no gross or

histologic changes in rats attributable to the test substance. Effects observed in dogs at 1.5 mg/L and higher included hypoactivity, reduced body weight, increased liver weight, yellow discoloration of aorta, hardened livers, and, in the high dose group, histologic changes of the liver, bone marrow, kidney, and testes. Seven dogs in the mid- and high group were sacrificed *in extremis*.

In a chronic study, groups of 50 Fischer 344 rats or B6C3F1 mice/gender/group were dosed with 0, 30, or 60 mg/kg/day (male mice) or 0, 60, or 120 mg/kg/day (female mice, male and female rats). There were no toxic effects in male or female mice. Although decreased survival was observed in male mice it was not correlated with toxicity or body weight effects. Therefore there did not appear to be a causal relationship between chlorobenzene exposure and reduced survival. Tumors common in aged mice occurred with similar frequency in all groups and were not considered related to chlorobenzene exposure. In rats, survival was decreased in the high-dose group. The only tumor type to occur with increased frequency was neoplastic nodules of the liver. In addition, a papilloma of the urinary bladder (one each in the 60 and 120 mg/kg/day male rats) and a tubular cell adenocarcinoma in one female 120 mg/kg/day female rat were observed. These were rare tumor types that did not occur in vehicle or untreated control rats.

No data on potential developmental toxicity of fluorobenzene were available. However, several developmental toxicity studies have been conducted using the close structural analogue chlorobenzene. These compounds have similar log Kow values (2.19 and 2.64 for fluoro- and chlorobenzene, respectively), suggesting that maternal/fetal partitioning for these compounds is likely to be similar. Fluoro- and chlorobenzene are also metabolized by similar pathways, with *para*- and *ortho*-phenols as the major products (Koerts et al., 1997; Rietjens et al., 1993). Phenolic metabolites of both compounds are subsequently conjugated with glucuronic acid and excreted. These similarities suggest that chlorobenzene should serve as a suitable model for fluorobenzene with regard to prediction of developmental toxicity. This conclusion is supported by *in silico* analysis of fluoro- and chlorobenzene using TOPKAT (Health Designs Inc, Rochester, NY) and MultiCASE (MULTICASE Inc., Cleveland OH). TOPKAT predicted both compounds to be negative for developmental toxicity in mammals. Similarly, MultiCASE predictions using modules for rabbit, rat, and mouse teratogenicity were negative for both halobenzenes.

The developmental toxicity studies for chlorobenzene indicated that chlorobenzene was not a unique developmental toxin in either rats or rabbits. Pregnant female Fisher-344 rats were exposed to 0, 75, 210, or 590 ppm chlorobenzene in air for 6 hours/day on days 6-15 of gestation. The maternal NOAEL was 210 ppm. Maternal toxicity occurred at 590 ppm as evidenced by decreased body weight gain on gestation days 6-8 and increased absolute and relative liver weights at study termination on gestation day 21. The fetal NOAEL was also 210 ppm based on an increase in skeletal variations (delayed ossification of vertebrae centra and bilobed thoracic centra) at 590 ppm. These variations were indicative of a slight delay in skeletal development among the fetuses (mild fetotoxicity) at a maternally toxic dose. The incidence of malformations (collectively or individually) was not increased in any of the exposed groups.

In addition, two inhalation developmental studies on chlorobenzene were conducted in rabbits. Pregnant female New Zealand white rabbits were exposed to 0, 75, 210, or 590 ppm

chlorobenzene in air (first study) or 0, 10, 30, 75 or 590 ppm chlorobenzene in air (second study) for 6 hours/day on days 6-18 of gestation. The maternal NOAEL was 75 ppm based on significantly increased absolute and relative liver weight at 210 and 590 ppm at study termination on gestation day 29. In the first study, a few chlorobenzene-exposed fetuses exhibited visceral malformations which were not observed among concurrent controls. However, there was no dose-related increase in malformations and there was no increase in malformations in chlorobenzene-exposed groups in the subsequent study. In the second study there was a significant increase in litters with resorptions at 590 ppm, although this effect was not observed at any concentration on the first study, and was within the range of historical controls. Therefore the conclusion was that chlorobenzene did not have an embryotoxic effect on rabbits.

While no formal reproductive toxicity studies have been conducted on fluorobenzene, no reproductive effects were observed in testes or ovaries in a 28-day inhalation study in rats. A 2-generation reproduction study with chlorobenzene was conducted by the inhalation route. Thirty male and female Sprague-Dawley rats per group were exposed to 0, 50, 150, or 450 ppm (0, 234, 702, or 2105 mg/m^3) 6 hours/day, 7 days/week. There were no substance-related effects on mortality, body weights, or food consumption. Mating, fertility, and other reproductive parameters and pup survival appeared unaffected by treatment. Liver weights were increased at 150 ppm and above in both generations and also at 50 ppm in the second generation male rats. The authors stated that the biological significance of the increased liver weights for the 150 and 450 ppm females and 50 ppm males was unclear. Liver and renal changes at 150 ppm and above and testicular effects at 450 ppm were observed in both generations. The authors state that the relationship of these testicular changes to chlorobenzene exposure was unclear because there did not appear to be any increase in intensity and/or incidence of testicular lesions among F1 adults that had longer exposure. The no adverse effect level was 50 ppm for F0 and F1 rats and >450 ppm for F2 offspring.

Fluorobenzene was equivocal in the Ames test. Fluorobenzene was tested in a preincubation assay in *Salmonella* strains TA100, TA1535, and TA98, without metabolic activation and with rat and hamster liver activation; a positive result with hamster liver activation was observed in strains TA100 and TA1535. A second study was conducted with a wider range of doses where the chemical was tested up to the highest dose permitted by toxicity. Based on limited data, fluorobenzene was not clastogenic in a mouse bone marrow micronucleus test.

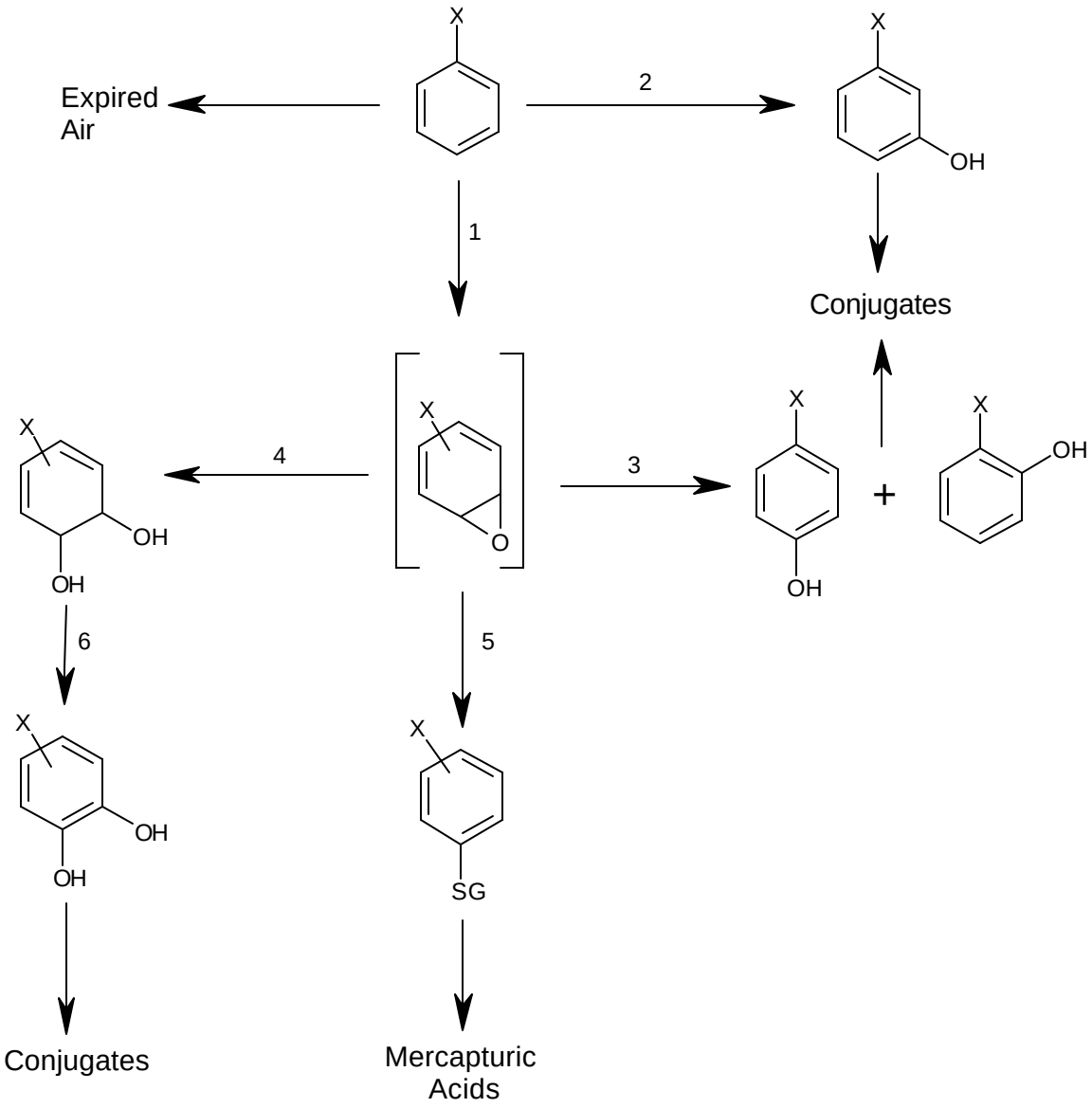
The following metabolism data is presented to support the use of monochlorobenzene as an acceptable surrogate compound to support the fluorobenzene database. The metabolism of monohalobenzenes has been the subject of extensive investigation for over 50 years (Yoshida and Hara, 1985; Krewet et al., 1989; Billings, 1985; Burka et al., 1983; Mills and Wood, 1953; Spencer and Williams, 1950; Kerger et al., 1988; Gut et al., 1996; Ogata et al., 1991; Ogata and Shimada, 1983; Koerts et al., 1997; Koerts et al., 1988; Rietjens et al., 1993). Qualitatively, monohalobenzenes, including chloro-, bromo-, iodo-, and fluorobenzene are all metabolized by common metabolic pathways (Figure 1). The initial step in halobenzene metabolism is hydroxylation at the C2, C3 or C4 position to form the corresponding 2-, 3- or 4-halophenol. Oxidation of halobenzenes to halophenols is though to occur via two mechanisms. Hydroxylation at C3 is thought to occur largely, if not entirely, by direct oxidation of the C3

position (pathway 2). Hydroxylation and C2 and C4 can occur through a direct oxidation of the appropriate carbon, but may also occur via formation of 2,3- and 3,4-epoxides, respectively (pathway 1). The epoxides undergo rearrangement through resonance stabilized cationic intermediates to form the corresponding 2- and 4-halophenols (pathway 3). The latter pathway accounts for the so called NIH shift seen with polyhalogenated benzenes (Koerts et al., 1998). For all halobenzenes, the major site for hydroxylation is the C4 position. The regioselectivity of oxidation of other carbon centers is driven by the Van der Waals radius of the halogen substituent. For larger halogens such as bromine and iodine, hydroxylation at C3 is favored over C2, while for smaller halogens such as fluorine the opposite is true. This is consistent with the findings of Burka et al., 1983 who demonstrated an inverse relationship between the size of the halogen substituent and the extent of hydroxylation at C2 for a series of monohalobenzenes. The involvement of epoxide intermediates is further supported by the identification of dihydrodiols (pathway 4) as urinary metabolites of chloro- and bromobenzene in rats, and *in vitro* following incubation of rat hepatocytes with halobenzenes (Billings, 1985; Zampaglione et al., 1973). The dihydrodiols thus formed can be oxidized by dihydrodiol dehydrogenase to form the corresponding catechols (pathway 5). Catechol formation is a relatively minor metabolic route in rats treated with chlorobenzene and fluorobenzene. However, 4-chlorocatechol is the major urinary metabolite of chlorobenzene in humans, suggesting that 4-fluorocatechol is likely to be a major human metabolite of fluorobenzene. These data are consistent with the relative expression levels of microsomal epoxide hydrolase in rats and humans. A final pathway for metabolism of halobenzene epoxides is conjugation of the epoxide moiety with glutathione (pathway 6). This reaction is accompanied by loss of water to form the S-halophenyl glutathione conjugate, which is excreted as the corresponding mercapturic acid in the urine.

In rats, approximately 20% of the administered dose of chlorobenzene is recovered as urinary metabolites within 24 hrs, with up to75% of the dose exhaled in the expired air (Krewet et al., 1989). Of the urinary metabolites, approximately 3% of the administered dose is recovered as phenolic metabolites, 11% as catechols and about 5% as mercapturic acids. The balance of urinary metabolites is composed of dihydrodiols and trace quantities of dehalogenated metabolites. Fluorobenzene metabolism in rats has been investigated using ^{19}F NMR, with emphasis on hydroxylated metabolites (Koerts et al., 1997; Koerts et al., 1998; Rietjens et al., 1993). These studies indicate that approximately 80% of the administered dose is recovered as fluorinated urinary metabolites. Of these, approximately 50% of the administered dose is accounted for by monophenols, with 2-, 3-, and 4-fluorophenol present at a percent ratio of 33:20:47. The remainder of the urinary metabolites was composed of catechols, and presumably mercapturic acids, though these were not explicitly identified and quantified. The proportion of the administered dose recovered as catechols was not reported, but based on NMR spectra presented, the urinary concentration of 4-fluorocatechol appears to be approximately a third of the 3-fluorophenol concentration. In addition, a trace amount of 2-fluorocatechol was also detected. Direct quantitative comparison of the results for chloro- and fluorobenzene is difficult, since both the route of exposure (i.p. versus gavage, respectively) and the dose levels (4.5 mmol/kg versus 0.5 mmol/kg) are different between the studies. Considering the high dose used in the chlorobenzene study (4.5 mmol/kg), saturation of metabolic pathways may account for the lower proportion of the administered dose of chlorobenzene recovered as urinary metabolites. Consistent with this hypothesis is a second study of chlorobenzene metabolism in rats, in which approximately 32% of a 2 mmol/kg dose was recovered as urinary metabolites.

Overall, the pathways for metabolism of halobenzenes, including chlorobenzene and fluorobenzene are essentially identical. Although directly comparable quantitative studies could not be found for evaluation, the trend in the available data suggests that following comparable doses, the extent of flux through the various pathways is likely to be similar for both chloro- and fluorobenzene.

Figure 1. Metabolic Pathways for Monohalobenzenes.



Test plan: A hydrolysis test using OECD Guideline 111 is recommended.

Human Exposure Information

(May 4, 2004)

Fluorobenzene is received at a contract manufacturing site in isotanks that are delivered from ocean freight ports of entry into the U.S. It is pumped directly from the isotank into steel tanks in a diked area prior to use in production. This is a closed system. It is then pumped from the storage tank into the reactors for processing. This is also a closed system. At the end of the processing, the fluorobenzene has been consumed. During the purification of the product(s), any residual fluorobenzene is recovered to closed drums and then recycled into the process. Fluorobenzene has some vapor pressure, and so fugitive fluorobenzene vapors from the process are captured by an aqueous scrubber. That fluorobenzene is recovered and recycled. All hoses, lines, and fittings are inspected prior to use, and drained and dried after use.

During these operations, operators wear appropriate personal protective equipment (PPE) to protect themselves from splash and vapor. All waste and byproduct liquids (including water) that might contain fluorobenzene are captured and disposed of at regulated, off-site treatment, storage, and disposal facilities (TSDF's) or publicly owned treatment works (POTW's).

Potential exposure may occur during unloading and processing when operators are measuring the volumes in the tanks and process equipment. There is also the potential for exposure when any recovered fluorobenzene is drummed and transferred into the tank after recovery. At these times operators wear appropriate PPE. There is also the potential for exposure to fugitive emissions during line breaking operations or in the event of equipment failure. Operators and maintenance personnel wear appropriate PPE during line-breaking and maintenance operations to protect themselves from splash and vapor.

PPE consists at a minimum of safety glasses with side shields, goggles (or face shield), gloves, coveralls, workboots, and respirators with organic vapor/acid gas cartridges. Additional PPE may also be required, depending upon other issues relevant to the operation being carried out. Safety showers, eyewash stations, and Self Contained Breathing Apparatus (SCBA) are available in close proximity to the operations area.

The contract manufacturer has procedures, practices, and controls in place to manage the risk of exposure and no incidents have been reported to DuPont. DuPont practices Responsible Care® and assesses the ability of a potential contract manufacturer to safely handle fluorobenzene prior to commencing a commercial relationship. This assessment includes reviews and audits of PPE, safety equipment and procedures, structural integrity, and safety practices.

The DuPont Acceptable Exposure Limit (AEL) for fluorobenzene is 25 ppm (8- and 12-hour TWA). No other limits have been established. Air monitoring has been conducted on fluorobenzene and results are shown in the table below.

EXPOSURE DATA

No. of Results	Exposure period TWA (ppm)	8-hour TWA (ppm)	Min. of Results (ppm)	Max of Results (ppm)
19	0.69	0.69	0.1	5.9

While DuPont handles fluorobenzene as a closed system intermediate, DuPont cannot guarantee that all fluorobenzene users maintain the same level of controls. This robust summary contains the developmental toxicity data for the structurally related compound, chlorobenzene to meet the developmental toxicity endpoints.

References for Summary:

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TEST PLAN FOR FLUOROBENZENE

Fluorobenzene **CAS No. 462-06-6**	**Data Available**	**Data Acceptable**	**Testing Required**
Study	**Y/N**	**Y/N**	**Y/N**
PHYSICAL/CHEMICAL CHARACTERISTICS			
Melting Point	Y	Y	N
Boiling Point	Y	Y	N
Vapor Pressure	Y	Y	N
Partition Coefficient	Y	Y	N
Water Solubility	Y	Y	N
ENVIRONMENTAL FATE			
Photodegradation	Y	Y	N
Stability in Water	N	N	Y
Transport (Fugacity)	Y	Y	N
Biodegradation	Y	Y	N
ECOTOXICITY			
Acute Toxicity to Fish	Y	Y	N
Acute Toxicity to Invertebrates	Y*	Y	N
Acute Toxicity to Aquatic Plants	Y**	Y	N
MAMMALIAN TOXICITY			
Acute Toxicity	Y	Y	N
Repeated Dose Toxicity	Y	Y	N
Developmental Toxicity	Y**	Y	N
Reproductive Toxicity	Y**	Y	N
Genetic Toxicity Gene Mutations (in bacterial cells)	Y	Y	N
Genetic Toxicity Chromosomal Aberrations (in *in vivo* micronucleus test)	Y	Y	N

* 24-hour data were available for the test chemical and 48-hour data were available for an analog chemical.

** Data were available on an analog chemical.

ROBUST SUMMARY FOR FLUOROBENZENE

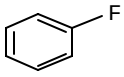
Existing published and unpublished data were collected and scientifically evaluated to determine the best possible study or studies to be summarized for each required endpoint. In the spirit of this voluntary program, other data of equal or lesser quality are not summarized, but are listed as additional references at the end of each appropriate section, with a statement to reflect the reason why these studies were not summarized.

1.0 Substance Information

CAS Number: 462-06-6

Chemical Name: Benzene, fluoro-

Structural Formula:



Other Names: Fluorobenzene
Monofluorobenzene
Phenyl fluoride

Exposure Limits: DuPont Acceptable Exposure Limit (AEL): 25 ppm (8- and 12-hour TWA)

2.0 Physical/Chemical Properties

2.1 Melting Point/Freezing Point

Value:	-40°C
Decomposition:	No Data
Sublimation:	No Data
Pressure:	No Data
Method:	No Data
GLP:	Unknown
Reference:	Budavari, S. et al. (1996). The Merck Index, 12th ed., p. 4212, Merck and Co., Inc., Rahway, NJ.
Reliability:	Not assignable because limited study information was available.

Additional References for Melting Point:

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